Diseases of Sebaceous and Sweat glands

- Acne vulgaris
- Rosacea
- Rosacea-like disorders:
  1. Solid facial edema
  2. perioral-periocular dematitis
  3. Papulopustular Eruption
  4. Rosaciform dematitis
  5. Steroid Rosacea
  6. pityriasis folliculorum
  7. Haber's Syndrome
- Lupus Miliaris (acne agminata)
- Fordyce spots
- Miliaria (Sweat Rash)
- Bromhidrosis
- chromhidrosis
- Hyperhidrosis
- Anhidrosis
- Granulosis Rubra Nasi
- Fox-Fordyce disease

Rosacea

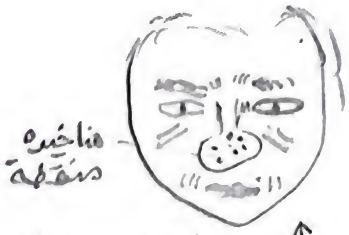
- **D.f**: chronic vascular inflammatory disorder - limited to: Center of face.
  ch.ch By: 1- persitant Erythema
  2- Telangectasia
  3- papules - pustules

- **Epidemiology:**
  - 30-50 yr - female more -
  - Incidence Highest in skin type I-II
    Lowest in darkly pigmented skin

- **Clinically:**
  1. affect mainly Center of face → cheeks - Chin - forhead - Nose (Blush area of face)
  2. Rarely may affect: arms - legs Chest - scalp - Scrotum (Red scrotum Syndrome)
  3. Skin → usually Oily

- **1ry - 2ry Features of rosacea:**

1ry	2ry
1- Flushing (Transient Erythema)	1- Buining-stinging (malar skin)
2- Non-Transient Erythema	2- Dry apperance (central facial skin)
3- papules - pustules	3- Soft or Solid facial edema
4- Telangectasia	4- Ocular manifistation
	5- peripheral location
	6- phymatous change



Types of phymatous rosacea

Rhinophyma (Fig. 52)	• Apparent initially as dilated patulous follicles at the distal end of the nose. • When marked, can lead to debilitating nasal deformity.
Gnathophyma	• Rare occurrence, with central chin typically involved. • May give rise to asymmetrical swelling.
Otophyma	• Usually affects the lower half of the helices & lobes of the ears.
Mentophyma	• Cushion-like, firm swelling of the central forehead.
Blepharophyma	• Swelling of the eyelids. • Usually seen as a component of edematous rosacea but may accompany severe papulopustular or ocular rosacea.

-Rosacea subtypes:

① Erythematotelangiectatic:
[vascular rosacea]

- **Blushing**
  - Recurrent
  - Then Long Lasting
  - eventually is permenant
- **Telangectasia**
  - Begin in the ala nasi
  - Then on nose-cheek
- **Edema**
  - Feeling of Fullness of Cheeks
  - Subtle induration of Cheeks
- **Central Facial edema**
  - persistant woody induration of Central Face
  - Respod well to Oral Isotretinoin
  - Should differentiate from Solid facial edema

② Papulopustular:
[inflammatory Rosacea] PPR

- varies from: Small papules-pustules to occasional deep peristant Nodules
- No comedones — No Scarring
- Papules are Asymptomatic

③ phymatous rosacea:

- Seen in Long lasting Cases in men
- it appears as: Red deep irregular Lobulated thickening of skin of the Nose with Follicular dilation
- It Represent:
  Hyperplasia of Sebaceous gland and connectic Tissue
- phymatous process May develop in extranasal sites as:
  Chin - forehead - cheeks - ears

④ Ocular rosacea

- Can occur without skin lesion. (Difficult to Diagnose)
  - Mild: Scaling of eyelid margin
  - More Active: • Blepharitis • • Conjunctival injections • Cysts from meibomian gland (chalizin)
    - Severe: • Keratitis • Uveitis • • Corneal neovascularization

- Rosacea Variants:

a) Granulomatous rosacea
- more persistant
- discrete
- red to red-Brown facial papules and Nodules

b) Pyoderma faciale:
(Rosacea fulminans)
- affect: Post-adolescent women
- 20-40 yr
- with NO previous History of Acne
- ch.ch By: Sudden erupted Purulent Nodulocystic lesions
- Specialy in: Face, NO Systemic $
- NO Comedone
- The Back, chest → Free.
- History of: Flushing
- treatment: Tetracycline (1g/day) or Isotretinoin (1mg/kg) with intralesional Steroids or liquid Nitrogen to the cyst
- Systemic Steroid Dose of 40-60 mg/daily tapered → over 3-4 weeks.
(This is one of the few times that oral steroid should be used in ttt of rosacea)

- Histopathology:

- ch.ch By: 1- Vascular Dilatation
2- perivascular non-specific inflammatory infiltrate
3- Solar elastosis
- in 10% of papular lesion: "Tuberculoid" picture Found < epithelioid cells / giant cells
- This lead to Diagnosis of: Rosacea-like Tuberculoid
- presence of granulomatous formation in rosacea is explained as: foreign Body reaction against keratinized cells of disintegrated hair structures or against elastic material.
- The granuloma may represent: Delayed Hypersensitivity Reaction to mite Demodex folliculorum

3

- Dermoscope:

1- Dilated Vessels
2- Prominant telangectasia
3- Large Polygonal Vascular Net.

Pathogenic hypotheses of rosacea

Aberrant innate immune system	• The innate immune response protects against microbial infection without requiring specific recognition of the pathogenic stimulus. • Activation of innate immunity leads to release of cytokines & antimicrobial molecules such as the peptide cathelicidin. • In rosacea, there is upregulation of cathelicidin and its processing serine protease, suggesting dysfunction of the innate immune system.
Ultraviolet radiation	UVR exposure has been shown to: • Induce angiogenesis. • Increase production of reactive oxygen species, which then leads to upregulation of matrix metalloproteinases (MMPs result in damage to blood vessels and the dermal matrix).
Vascular changes	• An increase in blood flow is seen within affected skin in rosacea. • Rosacea patients flush more readily in response to heat as compared with controls. • An elevated expression of VEGF & lymphatic endothelial markers is observed, suggesting stimulation of blood vascular & lymphatic endothelial cells.
Epidermal barrier dysfunction	• Increased transepidermal water loss. • Lowered threshold for skin irritancy. • Seen in both erythematotelangiectatic and papulopustular rosacea.
Microbes	• Demodex mites (folliculorum & brevis), which are commensals of normal skin, are found in greater numbers in rosacea patients. • Demodex infestation is associated with an intense perifollicular infiltrate of predominantly CD4+ helper T cells. • Other microorganisms which could be involved in the pathogenesis of rosacea: Staphylococcus epidermidis, Helicobacter pylori, Bacillus oleronius & Chlamydophila pneumoniae.

- Proposed role for micro-organisms in rosacae.

- **Step I:-**
  - unaffected rosacea-prone skin has ↑↑ expression of microbe sensing machinery and nomal flora
- **Step II:**
  - Commonsal agents may act as trigger Factors for rosacea-inflammation Through activation of pattern recognition receptors (PRR) or altered tolerance.
- **Step III:**
  - physiological and inflammatory changes in rosacea skin → Create a Micro-environment → suitable for altered growth or metabolism of microbes
- **Step IV:**
  - alteration in Commensal flora or
  - introduction of non-Commonsal agents → activation of inflammatay pathway.
- **Step V:** Immune Response that Result in clerance of excageibeiing agent →

-Exacerbating Factors:

1- Sun exposure
2- Alcohol
3- Tea - Coffe : it is the temprature of the coffe - tea Not The Caffeine
4- Psychic Factors

-Treatment:

• General •

1- avoid extreme Hot - Cold
   - → excessive sunlight
   - → Spices
   - → Hot liquids . Alcohol

• Systemic •

1- Tetracyclines:

- 250 mg / 3 times / daily
- 3-4 weeks
- 2nd generation Tetracyclines are effective → Minocycline / Doxycycline

2- Erythromycin: (200-250-500 mg)

- 30-50 mg/kg / day
- 2nd generation Macrolides → azithromycin / Clarithromycin

3- Metronidazole: Flagyl

200 mg/once or twice daily For 4-6 weeks

4- Isotretinoin: (Accutane) 10-40 mg/daily

• Topically •

1- Metronidazole → 0.75%. - 1%. Cream - gel - Lotion
2- Sodium Sulfacetamide → 10%. Cream - Foam - lotion - wash
3- Azelaic acid → 15%. gel
4- Brimonidine → 0.33%. gel (Mirvaso)
   - For: persistant nontransient facial Erythema of rosacea in Adult 18 yr or older.
   - Brimonidine tartrate → Highly selective α-2-adrenergic receptor agonist with Vasoconstrictive activity → In tt of Glaucoma
5- Ivermectin → 1% Cream (Soolantra) for tt of inflammatory lesions of rosacea.

6- Pimecrolimus → 1% cream twice-Daily

7- Tacrolimus → twice daily with 100 minocycline/twice

8- Benzoyl peroxide/ Clindamycin :-

• Plastic Surgery for Rhinophyma •

- Electrodessication OR Tunable dye laser for telangectasia

• Laser-Light therapies •

① Vascular laser Therapy:

- The standard pulsed Dye laser
- The Long-pulsed dye laser
- The diode-pumped frequency doubled Laser

② Intense pulsed-Light therapy

③ CO_2 laser: For phymatous Rosacea.

④ Nd-YAG laser + PDL :- Recently proved to be safe and effective in ~~th of~~ → Erythematato-telangiectatic rosacea. and ~~th~~ in the concentration of substance p → implicated in Pathogenesis of rosacea.

• Facial skin care in rosacea patient •

① wash with lukewarm water ② use soap-free cleansers
③ cleanser are pH Balanced and applied gently e' finger tips
④ Use sunscreen with Both UVA + UVB protection and SPF > 15
⑤ Sun Blocking creams :- Containing the physical Barrier: titanium dioxide or/and zinc oxide
⑥ use cosmetics and sunscreens that contain protective Silicones
⑦ water-soluble facial powder contain :- inert green pigment → helps to neurtralize the percoption of Erythema
⑧ Moisturizers :- Containing humectants e.g: glycein. occlusives: e.g: petrolatum → help Repair Epidemal Barrier
⑨ Avoid → astringents - toners - abrasive exfoliators
⑩ Avoid → Cosmetic that contain alcohol, menthols, Camphor, witch hazel, fragrance, peppermint
⑪ Avoid → Waterproof Cosmetics and heavy foundations That's difficult to Remove e'out irritations

Treatment of rosacea

Type of rosacea	Topical	Systemic	Other
Papulopustular	• Wide-spectrum antibiotics. • Tretinoin. • Metronidazole.	• Wide-spectrum antibiotics. • Isotretinoin. • Metronidazole.	• Pimecrolimus (1% cream) twice daily.
Erythemato-telangiectatic	Topical agents, e.g. azelaic acid, metronidazole & topical oxymetazoline (selective α1-agonist).	• Propranolol. • Clonidine.	• Laser.
Ocular	• Wide-spectrum antibiotics. • Cyclosporine 0.5% ophthalmic emulsion. • Metronidazole gel.	• Wide-spectrum antibiotics.	• Liquid paraffin eye ointment. • Eyelid hygiene & artificial tears.
Rhinophyma	-	• Isotretinoin May reduce nasal volume and halt the progression of rhinophyma.	• Surgical / laser remodeling.
Pyoderma faciale	-	• Oral steroids • Isotretinoin.	-

- Emerging therapies in Rosac :

I) Systemic therapy :

↳ ⓐ Sub-antimicrobial-dose **Doxycycline** :-
(20 mg / twice/day or once daily 40 mg)
act Through :

1- Down Regulation of Cytokines with Reduction of Neutrophils infiltration and deactivation of Downstream inflammatory pathway

2- inhibition of nitric oxide & Reduction of vasodilatation and cessation of Capillary wall degradation → Reduce leakage

3- Reduction of level of reactive Oxygen species → Slow destruction of Connective Tissue

4- inhibition of matrix Metalloprotienase

↳ ⓑ **B-Blockers** : act on flushing :-

1. Blocking the B-adrenergic Receptors on smooth muscles of Cut. Blood vessels. → vasoConstriction

2- Reduce anxiety, tackychardia → which can excagerate flushing reactions

Carvedilol → newer nonselective B-Blocker & marked antioxidant and anti-inflamatory action.

↳ ⓒ **Oral Ivermectin** :

Single Dose - oral ivermectin → microfilaricide → used in immunocompromised pts & rosacea like demodicidosis.

II) Topical therapy :

- all are Targeted to control of Demodex Folliculorum and D. brevis mites.
- Permethrin 5%. - Crotamiton 10%.
  Ivermectin 1%

- Rosacea-like disorders:

[1] Solid facial edema (Morbihan's disease)

[2] perioral - periocular dermatitis :-

- in young females
- lesion: Persistant Erythematous Eruption of tiny papules and pustules → around the mouth, nose, eyes.
- a clear Zone → seen around the vermilion Border of lips
- etiology:

- Due to prolonged therapy ē fluorinated Steroids - Sunlight - Oral cps
- fluorinated tooth paste
- heavy users of cleansers, moisturizers.

- treatment:

1. Stop → The topical corticosteroid Therapy
2. Systemic antibiotics: tetracycline Doxycycline - Minocycline - Erythromycin Azithromycin → 4 - 6 weeks
3. Topical Metronidazole or Azelic Acid
4. pimecrolimus Cream 1% → twice daily

[3] papulopustular Eruptions :-

Due to → epidemal growth factor Receptor EGFR inhibitors

[4] Rosaceiform dermatitis :-

- facial Erythema - small papules - numerous pustules
- caused By: application of topical Calcineurin inhibitors on face.

[5] Steroid Rosacea:

- florinated and potent corticosteroids Cause problems (topical or inhaled) → induce and excagerbate Rosacea.
- presence of → Rosacea - like lesion on the upper Lip and around ala nasi

↓

Clue that corticosteroid involved.

[6] pityriasis follicularum :-

- Roughened - whitish - scaling Skin surface
- superimposed on a Background of faint Erythema + scattered fine papules and pustules
- young - middle-age women
- Skin scraping → reveals presence of - Demodex mites

[8]

- application of → Topical sulfur preparation OR permethrin 5% at night ~~for~~ 2 weeks → ttt

[7] Haber's Syndrome:-
- rare familiar Genodematosis
- Ch.ch By: Early Onset facial Erythema - Verrucous papules Truncal Keratotic lesions and pitted atrophic scars.

• Lupus Miliaris Disseminatus Faciei • (Acne agminata)

LMDF

- Lesion:
Discrete - Redish papules
site: Face mainly on Eyelids cheeks, upper Lip.
- Erythema - telangectasia → The ch.ch of Rosease is Absent
- involute spontaneously in a year → leaving:- pitted small scars

[9]

- In 2000 → The name changed :- from LMDF to FIGURE :
facial idiopathic Granulomatous è Regressive Evolution
- Histopathology:
- Large tubercles → composed of:
  - epithelioid cells
  - giant cells → large areas of Caseation necrosis in the Center
- at the periphery: inflammatory infiltrate
- Histogenesis:
No evidence to support tuberculous etiology
Dosn't Respond to anti-Tuberculous Drugs
- treatment:
Long term therapy è • Tetracycline • minocycline OR • Isotretinoin-

• Fordyce Spots •

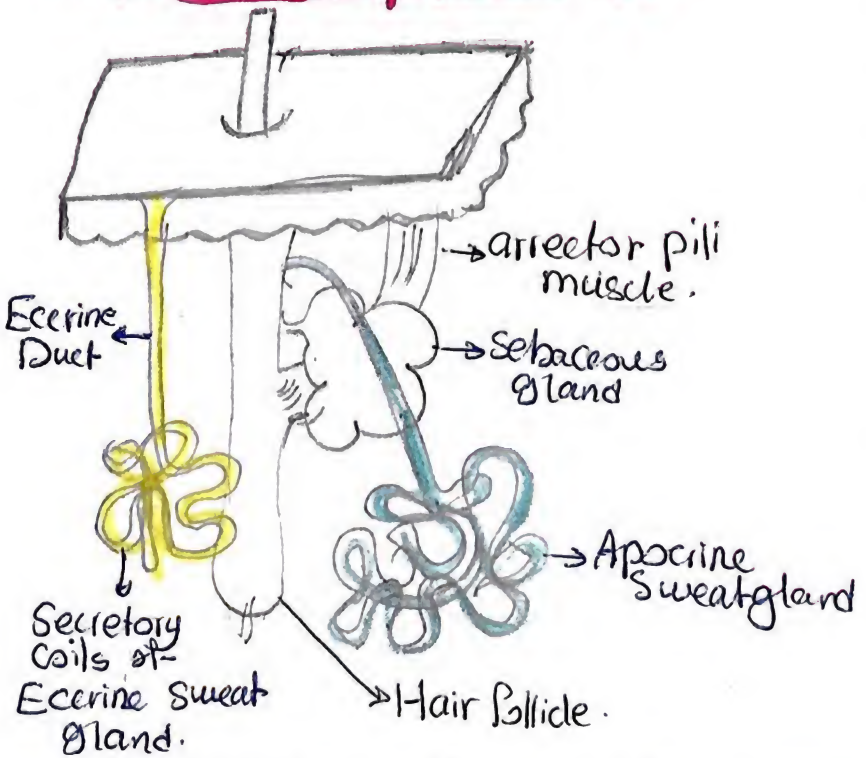
- D.F: heterotropic Sebaceous glands that can occur:
  - Around the vermillion Border of the lips or within the oral mucosa ; Asymptomatic - multiple symmetrical - barely elevated - Yellow papules
  - ventral surface penile shaft → inflamed
  - Areolar area of the Breast
- treatment: → Oral Isotretinoin → for extensive lesions
  → Carbon Dioxide ablative laser

Sweat gland

- **2 main types:** + 3rd type

Eccrine | Apocrine | apoecrine

Anatomy - physiology:



- Humans have upto 4 million sweat glands over all Body.
  3 millions are Eccrine sweat glands
- Ratio 1:1 = apocrine to Eccrine → in axilla 1:10 in elsewhere

	Eccrine sweat glands	Apocrine sweat glands	Apoecrine sweat glands
Localization	• Entire body skin (except the external auditory canals, vermilion lips, clitoris & labia minora). • Highest density on palms & soles.	• Axillae, anogenital, periumbilical, nipples.	• Axillae.
Embryology	• From downgrowth of epidermis at 3rd month of IUL independent of pilosebaceous follicle. • No new glands develop after birth.	• As a part of pilosebaceous follicle in 4th of 5th month of IUL	• Not present at birth.
Morphology	• Long, thin duct opens to skin surface. • Secretory coil with narrow lumen.	• Short, thick duct opens into upper part of follicular canal. • Secretory coil with wide lumen.	• Duct comparable to eccrine gland duct. • Secretory coil with dilated & non-dilated tubular segments.
Cell types in secretory coil	• Large secretory clear cells, dark cells, and myoepithelial cells.	• Epithelial & myoepithelial cells.	• Eccrine & apocrine morphologic features.
Secretion	• No cellular breakdown, i.e. merocrine glands where the cells are not destroyed during secretion.	• Pinching off parts of apical cytoplasm → oily odorless fluid (bacterial decomposition → odors).	
Main innervation/ neurotransmitter	• Sympathetic fibers/acetylcholine.		
Development	• Present at birth. • No relationship to pilosebaceous follicle.	• Present at birth. • Associated with terminal hair follicle.	• Develop during puberty from eccrine-like precursor cells. • No relationship to pilosebaceous follicle.
Function	• Thermoregulation/role in hyperhidrosis and hypohidrosis.	• Unclear/some role in olfactory communication. • Role in follicular apocrine Fox-Fordyce disease	• Thermoregulation/role in axillary hyperhidrosis & non-follicular apoeccrine Fox-Fordyce disease.
Composition of the secretions	Odorless clear fluid: • pH 4 – 6.8 (hypotonic). • H_2O, Na, Cl, K, urea, lactate & glucose.	Sterile, odorless and viscous: • pH 5.0 - 6.5. • Precursors of odoriferous substances (cholesterol, triglycerides, fatty acids, cholesterol esters, squalene). It also contains androgens, carbohydrates, ammonia & ferric iron.	• Unknown.

Miliaria (sweat Rash)

1- D.F:

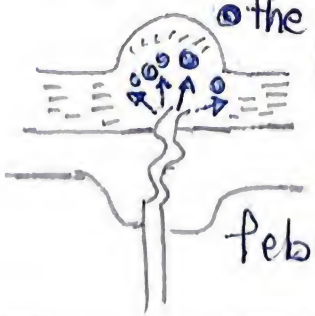
- Common condition after prolonged exposure to Hot-humid environment: tropics. Occupation
- most common in: neonates → (whose eccrine sweat ducts Not fully developed)

2- Pathogenesis:

- excessive sweating
- under occlusive clothing lead to → maceration of the Stratum corneum.
  ↓
  Sufficient to cause Blockage of eccrine Duct
- followed By → Rupture of The Duct and sweating below the level of obstruction

3- Clinically: according to level of obstruction

① Miliaria Crystallina:



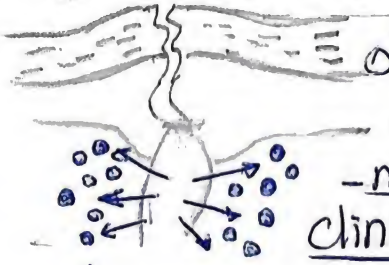
- the obstruction is in Horny Layer → Subcorneal vesicle formation
- occure: Sunburn
  Febrile illness

- associated: profuse sweating, in newborn e: excessive clothing and heavy sweating
- clinically:
  Asymptomatic - Small - clear - non inflammatory Thin walled Vesicles
- mainly on: Trunk
- Vesicles → Rupture → superficial branny desquamation

② M. ruba:

- the obstruction: in the intraepidermal Sweat Duct → Spongiotic Vesicle in stratum malpighii
- it develop in: area of friction with clothing in flexures and after occlusive therapy
- clinically:
  - Small. papulovesicles Surrounded by Erythema with pricking sensation
  - may be pustular (pustulosa) → in chronic extensive cases
- its Sterile bacteriologically

③ Miliaria Profunda:



- obstruction occur:
  - at DEJ.
  - mainly in: Tropics

clinically:

Asymptomatic • pale • firm papules mainly on: Trunk

lesions:

Asymptomatic because sweat Retention vesicle that forms → lies in the upper dermis Below the level of the Itch Receptors

-Complications:

① 2ry bacterial infection → miliaria pustulosa

② Disturbance of heat regulation → when Large numbers of glands are occluded and non-functional → these individuals at Risk for significant thermoregulatory Problems and Compensatory Hyperhidrosis of the Face.

Type	Location of obstruction	Cutaneous lesions	Patient	Common locations
Miliaria crystallina	Stratum corneum.	Non-pruritic, clear, fragile, 1 mm vesicles.	Neonates <2 weeks of age. Children & adults in hot climates.	Face & trunk.
Miliaria rubra	Mid-epidermis.	Pruritic, erythematous, 1-3 mm papules; may have pustules.	Neonates 1-3 weeks of age. Children & adults in hot climates.	Neck & upper trunk.
Miliaria profunda	Dermal-epidermal junction.	Non-pruritic, white, 1-3 mm papules.	Adults in hot climates; often with multiple bouts of miliaria rubra.	Trunk & proximal extremities.

③ Axillary and inguinal Lymphadenopathy → seen

④ "tropical anhidrotic asthenia" → applied to These pts

-treatment:

1. avoid excessive Sweating. Stay in AC.
2. wearing Light clothing
3. Gradually → The obstructed sweat Ducts Shed their occluding Keratinous plugs → normal sweating
4. Anhydrous Lanolin → prevent Ductal Blockage. allowing sweat to flow to the skin surface.
5. Occlusive ointments → avoided
6. Calamine Lotion → for Cooling Symptomatic relief after miliaria develops.

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Diseases of Sweat gland:

1- specific diseases of Sweat gland:
- Miliaria
- Altered composition of ecrine Sweat
- Cytologic inclusions and other morphologic changes of Diagnostic importance in sweat gland.

2- Hyperhidrosis

3- Hypohidrosis + Anhyidrosis.

4- Sweat as a predisposing influence on Skin Disease

5- Apocrine sweat gland Disease

Altered Composition of Eccrine:-

1 Altered electrolyte Content: systemic disorders that affect the levels of electrolytes in sweat.

↑↑ level of electrolytes in sweat:
- Cystic fibrosis: ↑ CL (>60 mEq/L)
- Endocrine disorders: - Adrenal insufficiency
  - Myxedema
- Malnutrition

↓↓ level of Electrolytes:
- endocrine disorders:-
  - Aldosteronism
  - Cushing's Syndrome
  - Thyrotoxicosis
- Hypoproteinemic edema

2 Bromhidrosis:-

Apocrine bromhidrosis
- Axillary: penetrating odor
D.t → Bacterial decomposition of apocrine sweat mainly:- Axilla > e` puberty
- The apocrine sweat: originally odorless, apart of excreted substances as garlic.
- Androgens: of apocrine sweat → gives rise to odoriferous substances (pheromones)

Eccrine bromhidrosis

Keratogenic
- Pyo maceration of St. Corneum with bacterial degradation of Keratin
- plantar or Interliginous

Metabolic
- phenylketonuria (musty-Mousy odor)
- Maple syrup Urine disease (Sweat odor)

Exogenous
- Foods: - garlic - Curry asparagus
- Drugs:- penicillin - bromides
- Chemicals: dimethyl sulfoxide

● Treatment of Bromhidrosis :-

1- Frequent washing of axillary region
2- ↓↓ Foods as garlic from Diet
3- Local: Antibacterial substances aluminium salts
4- frequency-doubled, Q-switched Nd:YAG laser → effective
5- Botulinum toxin A → future therapy

3 Chromhidrosis :-

- Apocrine in origin
- Found in: genital - axillary - areolar facial skin
- chromhidrosis Reported only: on The face, axillae, breast areola.
- lipofuscin pigment → responsible for the colored sweat.
↓
This pigment is produced By Apocrine
- The various Oxidative states → Responsible for the ch.ch → yellow green - Blue - Black secretion observed in apocrine chromhidrosis

- Eccrine Chromhidrosis: is Rare, occur with: Digestion of Certain dyes or Drugs :- Red sweat in Pts receiving Clofazimine - Rifampicin.
- Pseudo-chromhidrosis: when clear eccrine sweat become colored on the surface of the skin

Cytologic inclusions & other morphologic changes of diagnostic importance in the sweat glands

Disorder	Microscopic changes
Hypothyroidism	PAS-positive, diastase-resistant granules in secretory cells.
Lymphoma – Multiple myeloma – Heat stroke	Vacuolar degeneration of secretory cells or ducts.
Mucopolysaccharidoses	Vacuoles in secretory cells.
Sphingolipidoses	Lipid inclusions in secretory cells.
Trisomy 13, 18 or 21	Differences in duct: Secretory coil length.
Severe acute respiratory syndrome (SARS)	Viral particles in lumen of sweat gland tubule.
Uremia	Reduced size of sweat glands.

Hyperhidrosis

- **D.F:** excessive production of eccrine sweat.
- **Types:**
  - Primary (idiopathic)
  - Secondary
    - medication
    - Disease.

1ry hyperhidrosis

- most common Type
- Volar or axillary
- Diagnostic Criteria:
  - Focal, visible, excess sweating
  - present at least for 6 mnths
  - No apparent 2ry Causes
  - At least 2 of the following:-
    1. Bilateral - symmetrical
    2. impaire activity of daily life
    3. at least one episode/week
    4. Age of onset < 25yr
    5. +ve family History
    6. Stop During sleep.

- Axillary hyperhidrosis is D.t: overactivity of eccrine glands Unlike axillary odor → mainly Apocrine in origin
- palmoplantar Hyperhidrosis :- may be inherited in autosomal dominant manner

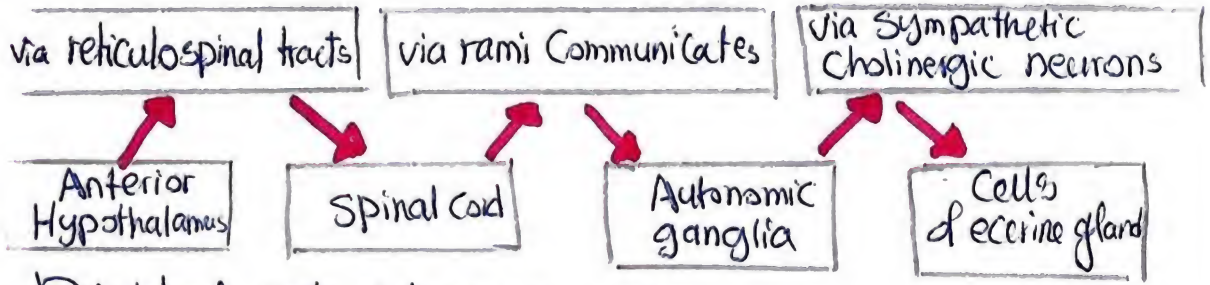
Volar (palmoplantar) hyperhidrosis (Fig. 1)	Axillary hyperhidrosis
50-60%	30-50%
Onset is during childhood.	Onset is at or soon after puberty.
Entire palm and sole, the lateral aspects, tips and distal dorsal skin of the fingers.	Right axilla usually produces sweat >left.
	Odor (axillary bromhidrosis) is usually absent.

Both types may coexist.

2ry Hyperhidrosis

- Localized
- Generalized

- Neural impulses for sweating :-(Sudomotor impulses)



- Divided into categories:- Based on the source of The neural impulse Driving the response :
  - Cortical
    - Hypothalamic
  - medullary
    - Spinal Cord
  - Local
    - non-neural in origin

1 Cortical:

- Isolated sweating of palm-soles
- Due to: Cortical excitation By:
  - emotional
  - sensory stimuli
- several disorders of Cornification and other genodermatoses affecting palmoplantar Skin are associated with Volar hyperhidrosis:
  - PPK
  - epidemolysis bullosa simplex
- Rarely Hereditary autonomic Neuropathies → Can also exhibit Cortical Hyperhidrosis

2 Hypothalamic (thermal)

- The hypothalamus Responsible for → Thermoregulation.
- elevation temprature of Body Blood By 0.5 F or more
  ↓
  excites the hypothalamic nuclei
  ↓
  vascular Dilation + generalized sweating
- it may also occur Due to: Direct hypothalamic Stimuli e.g
  - pheochromocytoma
  - medications
  - Toxins.
- Maybe → Nocturnal OR → Diurnal

3 Medullary: (Gustatory)

Taste Receptors are the source of the afferent impulses that stimuli Sweating

physiologic

- Triggered By:
  - Spicy foods
  - alcohol
  - Citrus fruits
- Mediated Through: Reflex arc impulses from taste bud Receptors travel via glossopharyngeal nerve to nuclei within The medulla oblongata
  ↓
  leads to provocation of sweating + Erythema on: upper lip Cheeks Rather Than The usual Response of Salivation
- Familial tendency
- No other Concurrent Local or Systemic signs or symptoms

pathologic

- occur when disrupted nerves for sweat → Connected nerves for Salivation.
- Caused By:
  - injury or disease of parotid gland (Frey's Syndrome)
    - Trauma to Sympathetic Trunk
  - CNS disorders:
    - encephalitis
    - Syringomyelia

4 Spinal Sweating :- (Cord transection)

- Spinal disorders Result in:
  - Lack of thermal Sweating Below the injury
  - Unusual patterns of Hyperhidrosis
- Bouts of "mass reflex Sweating" →
  - ↳ initiated in the skin Below the level of the Cord interruption.
  - ↳ most intense in skin segments Close to the level of the Transection
  - ↳ Mass reflex Sweating → doesn't occur in transection below T8 - T10.

5 Autonomic Dysreflexia :-

- Direct Stimulation
  - e.g.: electrical - physical
  - Drug induced.
- → Sympathetic axon → Cause Sweating
- → may be seen with inflammatory Skin disease

6 Compensatory :-

- Anhidrosis in one area → Cause hyperhidrosis in another
- Miliaria - Diabetic neuropathy - Sympathectomy → well-known Causes

7 Non-neural :-

1- Direct Stimulation of eccrine glands → Cause Sweating
2- Triggers include:
- → Local heat
- → Drugs :- Acetylcholine - Insulin
- → Vascular Tumors

Causes of generalized hyperhidrosis	Causes of localized hyperhidrosis
• Febrile infective illnesses: TB, malaria, brucellosis, etc. • Metabolic diseases: DM, hyperthyroidism, hyperpituitarism, hypoglycaemia, pheochromocytoma. • Menopause. • Underlying solid malignancy and lymphoma. • Parkinson's disease. • Congestive heart failure. • Neurological disorders. • Peripheral neuropathies, brain diseases, ... • Episodic hypothermia with hyperhidrosis. • Generalized hyperhidrosis without hypothermia. • Drugs: e.g. Fluoxetine.	• Spinal cord injury. • Intrathoracic neoplasia. • Gustatory hyperhidrosis. • Functional and true sweat gland nevi. • Sweating associated with local skin disorders: o Glomangioma. o Blue rubber bleb nevi. o Pachydermoperiostosis. o Pretibial myxoedema. o POEMS syndrome. o Burning feet syndrome. • Compensatory. • Idiopathic unilateral circumscribed hyperhidrosis.

Drugs that Can Stimulate eccrine Sweating :-

1. Direct Acting cholinomimetic Agents
   - Acetyl choline
2. Cholinesterase inhibitor
   - Anti-Alzheimer's drugs
   - Anti-myasthenic
3. Adrenomimetic Agents
   - Dopamine
   - epinephrine
   - Norepinephrine
4. Anti-diabetic hypoglycemic :-
   - Insulin - Sulfonylurea
5. CNS Stimulants:
   - Amphetamines - Caffeine
6. Antidepressants:
   - MAOIs - SSRI - tricyclics
7. Anti-psychotics:
8. Antipyretics: NSAIDs
9. opioids

- Diagnosis :

1. Differentiate Between primary and secondary → History, Physical exam, Lab testing

Laboratory test	Disease
Serum electrolytes, BUN, creatinine	Renal disease (rare)
Blood glucose level	Diabetes mellitus
Thyroid function tests	Hyperthyroidism
Skin test for tuberculosis (e.g. PPD)	Tuberculosis
Chest X-ray	Tuberculosis, neoplasm
Complete blood count	Infection
Sedimentation rate	Infection, neoplasm, inflammatory disease
Antinuclear antibodies	Autoimmune connective tissue disease
Urinary catecholamines	Pheochromocytoma

2. The severity of the condition

	Palmoplantar hyperhidrosis	Axillary hyperhidrosis
Mild	Moist palm or sole without visible sweat droplets.	Sweat stains on clothing: 5-10 cm.
Moderate	Sweating toward the fingertips.	Sweat stains on clothing: 10-20 cm.
Severe	Drip sweat.	Sweat stains on clothing: >20 cm.

3. Colorimetric techniques as :- Starch iodine :- Iodine solution e.g (3.5% in alcohol) → applied to clean shaved skin → Dry → Then starch powder (Cornstarch) → The mixture Turn Blue-Black in site of Sweating
4. To document amount of sweat produced → Gravimetric technique → Evaporative measurement
   - Infrared thermography

Treatment of hyperhidrosis

	Therapy	Frequency & recommended dose	Side effects	Duration	Comments
First-line	*Topicals* • Aluminium chloride hexahydrate – 6.25% (e.g. Xerac AC®), 15% (with 2% SA), 20% (e.g. Drysol®). • Aluminium chloride – 12% (e.g. Certain Dri®). • Zirconium salts. • Aldehydes.	Use nightly for 3-5 nights, then every few days as needed.	• Burning. • Irritant contact dermatitis.	Days	• Blocks sweat ducts. • Aldehydes not recommended due to sensitization. • Zirconium salts may be effective in axillae, but not volar surfaces.
Second-line	*Iontophoresis*	2-3 times a week.	Discomfort during procedure.	Days	Blocks sweat ducts.
	Botulinum toxin A	Every 4-6 ms.	• Discomfort during injection • Weakness of underlying muscles.	Months	Prevents release of acetylcholine.
	Oral therapy	As needed		Hours	
	• Oxybutynin	• 1.25-5 mg bid	• Dry mouth & urinary retention most common; also confusion & decreased mental status.		• Anticholinergic
	• Glycopyrrolate	• 1-2 mg bid			• Anticholinergic
	• Clonidine	• 0.1-0.3 mg bid	• Hypotension, rebound hypertension.		• α2-adrenergic agonist.
	• Propranolol	• 10-40 mg bid	• Hypotension, bradycardia, hyperhidrosis with long-term use.		• β-adrenergic blocker.
	• Clonazepam	• 0.25-0.5 mg bid	• Sedation.		• Anxiolytic.
Third-line	*Local excision*	Once	Scarring	Permanent	Last resort
	Sympathectomy		• Compensatory hyperhidrosis. • Horner's syndrome.	Usually permanent	

bid, twice daily; SA, salicylic acid.

Botulinum Toxin type A :- (BTA)

- for the treatment of axillary hyperhidrosis :-

→ **Mechanism of Action:**

BTA prevent release of acetylcholine from Cholinergic neurons

→ **Effect:**

Injection into Hyperhydrotic skin will produce
→ near anhidrosis for 4-6 months

→ **Side effects:** (short-lived)

↳ Muscle weakness → of intrinsic muscles of the hands or feet → Resolve over 2-5 weeks

↳ Compensatory Hyperhidrosis Not observed

→ Both Botox and Dysport have similar received reduction of sweating in The treatment of primary axillary Hyperhidrosis

Using: Convesion factor of 1:3
(100 units Botox vs 300 units Dysport)
and primary palmar hyperhidrosis using Conversion Factor 1:4

→ Tt of axillary hyperhidrosis with B. toxin A reconstituted in lidocaine OR Normal

Saline → was descriped in a randamized Side-By-Side - double Blind study.

↓

- The Result were the same
- associated è significant reduced pain.

- Laser treatment of Hyperhidrosis

- photothermal destruction of sweat glands (desquamation and Rupture of sweat gland)
- Using: → Subdermal Nd:YAG laser safe and effective
- Sweat Reduction is proved to be stable over period of upto 24 mnths.
- treatment of axillary hair using 1064 Nd:YAG laser → Cause hyperhidrosis and That effect is Not Transient

- Microwave thermolysis

- Microvave technology → well suited for targeting Sweat glands -
- allowing protection for Both: upper skin layer and structures Beneath the S.C Fat.

3- Anhidrosis

- **D.F:** absence of sweat from the surface of skin, in the presence of appropiate Stimuli
- **Causes:**

Neuropathic
- Hysteria
- Disease of tumor:-
  - Hypothalamus
  - spinal cord
  - peripheral nerves

e.g: Leprosy - DM alcoholism. Sympathectomy

Sweat gland Disturbance
- Aplasia
- Congenital Ectodemal defect
- Ichthyosis
- Atrophy:-
  - Scleroderma
  - Acrodermatitis atrophicans
- plugging of sweat Duct:-
  - miliaria – lichen planus
  - psoriasis

Idiopathic
- Neonatal
- Sweat gland fatigue
- Idiopathic aquired anhidrosis

- **Sweat as predisposing influence on the Skin Disease:**

1- wet skin → more permeable than Dry skin
2- wet skin → more likely to develop Contact allergies than Dry skin

3- Several diseases are aggrevated By: Sweat:
- Hailey - Hailey Disease
- Grover's disease.
- Folliculitis
- pitted Keratolysis

Granulosis Rubra Nasi

→ Rare autosomal Dominant
→ Onset: During the first decade of life
→ Resolve: at pubeity
→ Clinically:
Hyperhidrosis of nose; Followed By Macular Erythema, Papules - Vesicles
- may extend to:- Cheeks, lips - upper chin

→ treatment:
Drying lotions. Reassurance

Diseases of Apocrine Sweat glands

- Fox-Fordyce disease: [Apocrine Miliaria]

- Occur mainly: mainly in women soon after puberty
- Ch·ch By: → the presence of Itchy - discrete firm, follicular papules dome shape - Skin colored in area which Apocrine glands are found
  - → Axillae
  - → areola
  - → anogenital Region
- The condition improve in:
  During pregnancy → Dit: ↓↓ apocrine gland Secretion.
- Pathogenesis + Histopathology :-
  - Hormonal disturbances → formation of a "Keratotic plug" in the distal Apocrine duct
  - Followed By: • Rupture of the Apocrine Duct.
    - Local microvesicle formation
    - paraductal Acanthosis follow.
  - inflammatory infiltrate → at dermis
- Treatment:
  1. Topical, intralesional Corticosteroid → 1st line
  2. Topical tretinoin
  3. Topical clindamycin (twice daily)
  4. Isotretinoin
     15-30 mg/day → 4 months
     - Short Term success
     - Condition Return 3 months after stop ↓↓
  5. Hormonal therapy in women
  6. UViolet light
  7. dermabrasion
  8. Surgical excision → non Curative

Acne

	Acne vulgaris	Lupus miliaris disseminates faciei
CP	- Comedones, papules, pustules, nodules, cysts - Face, chest, shoulders, upper back - Post-inflammatory erythema & pigmentation - May heal with scarring - Seborrhea	- Discrete, reddish papules - Face mainly eyelid, cheeks, upper lip - Absent erythema & telangiectasia - Involutes spontaneously with pitted small scars
HP	1- Comedone → keratinous debris, micro-organism, hair, sebum 2- papules → lymphocytic perifollicular infiltrate 3- Rupture of follicle wall → escape of contents → aggregation of neutrophils → pustules & nodules	1- Large tubercle → epitheliod cells & giant cell 2- Central caseation necrosis 3- Peripheral inflammatory infiltrate
Pathogenesis	1- Increased sebum production 2- Ductal hypercornification 3- Proliferation of P.acne 4- Inflammation	1- No evidence support tubercle etiology 2- It may be related to rasacea
ttt	Anti-acne agents	Tetracycline, minocycline, isotretinoin

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Acne

Neonatal acne	Infantile acne
- May be present at birth or develop during the 1st few months - More common in males - Mild & regress spontaneously by the age of 6 months - Not associated with significant scarring or increased incidence of acne later in life	- Begins between 3rd & 6th month - More common in males - Severe nodules & cysts & may persist to age of 5 years - Associated with significant scarring or increased incidence of acne later in life - Associated with vililizing tumors
Pathogenesis: - Species of malassezia - Response to topical ketoconazole - Sebum excretion: high level	- Intrinsic hormonal imbalance ↑ testosterone, LH, DHEA

أسأل كم الدعاء لي

Amylee

Acne vulgaris	Acneiform eruption
- Comedone, papule, pustule, nodules, cyst - Face, chest, shoulders, upper back - Age: male: 16-19, females: 14-16 - Chronic inflammatory disorder - Acne variants: .... - ttt: anti-acne agent	- Papule, pustule - Not confined to the usual sites - Passing acne age - Sudden onset - Types: ... - Resolve slowly with withdrawal of the cause

(18)

Q Acne:

1- Pathogenesis of acne vulgaris.
2- Give an account on acne vulgaris
3- Give an account on Acne variants.
4- Pathogenesis, clinical types & therapeutic modalities in acne vulgaris.
5- Compare between acne conglobata & acne fulminans.
6- Compare between Acneform eruptions & acne vulgaris
7- Compare between neonatal acne & infantile acne
8- Management and Medical ttt of nodulocystic acne.
9- Acneform eruptions.

10- Preoperative considerations of laser ttt of acne scar
11- Compare: AV & drug induced acne
12- Compare: AV & lupus miliaris disseminated faciei
13- CP & management of rosacea
14- Eye manifestations of rosacea
15- Ecrine & apocrine sweat gland: distribution, histological shape, location, mechanism of secretion, diseases affecting them
16- Hyperhidrosis, causes, clinical variants & ttt

acne

1) acne varint ?

   1. اهم واحدة acne fulminant clinically? Ttt?

2) classification of acne ?

   inflammatory and non inflamtory عاوز يسمع

   a. mild.mod.severe مش عاوز يسمع

3) Gram negative folliculitis

4) Isotretinoin cumulative dose مهم جدا

   امتى ندى اقل من ٠.٥ مجم فى اليوم فى الروزسيشيا

5) Acne fulminans and conglobata ?

6) Why is hemorrhagic nodul in acne fulminans ? vascular

7) Systemic maniefest of acne fulminans ?

8) Lupus milliaris ?

9) Compare Lupus milliaris and acne ?

10) pAthogenesis of acne ?

11) syndrome associated with acne?

12) Emergency of acne ?

13) Type of comedo of acne conglobata ?

14) Reason and ttt of an fulminans and conglobata?

15) Low dose uses of isotretinoin?

16) اكتب روشتة لحب الشباب ؟ ده سؤال روتينى ف الشفوى

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